et al.[1] More recently, I reviewed[2] in detail the practical consequences of these pH-dependent mechanisms on the stability and efficacy of glutaraldehyde hospital formulas. This study clearly showed that there were only two fundamental types of glutaraldehyde compositions: those described in the alkaline range by the Ethicon patent (expired in January 1978) and those described in the acid and neutral range by the Wave Energy Systems patents. Under normal hospital use the alkaline compositions have a short life (maximum 14 days) and their only theoretical advantage is a slightly faster sporicidal action at room temperature. No substantial cidal advantage over the acid formula has ever been found when dealing with non-sporulated bacteria, viruses, and mycobacteria. Unlike alkaline compositions, potentiated acid glutaraldehyde solutions such as Sonacide have a slow rate of polymerisation, which corresponds to a continuous use life in hospitals of 28 days. Thanks to this extraordinary stability Sonacide decreases considerably the cost of any hospital disinfecting procedure, as recently demonstrated by Ayerst Laboratories in a two-year study covering 32 North American hospitals.[3]

Tarnishing and superficial metal erosion can sometime occur with glutaraldehyde disinfecting solutions. As your correspondent (26 May, p 1425) noticed, the higher the pH the milder is the potential tarnishing problem in the 6-8 pH range. One should, however, recall that potential tarnishing occurs mainly with scratched, worn-out, or poorly plated instruments which have been overexposed to disinfectant solutions. Let us recall, for instance, that five years of nationwide use of Sonacide (pH 4-6·2) in the USA never brought to light any major tarnishing problem when the product was used according to the recommendations of the Environmental Protection Agency label.

The recent discovery[4] of the biocidal synergistic effect of ultrasonics on acid potentiated glutaraldehyde is another indication of the promising future of dialdehyde disinfecting techniques in hospitals.

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Abnormal cilia

SIR,—We were pleased to see your leading article on abnormal cilia (23 June, p 1663) and would like to draw attention to another area where cilial abnormalities have been reported, which may be of considerable medical significance.

We have described[1] abnormalities in nasal cilia in patients with Usher's syndrome and other forms of retinitis pigmentosa. In life direct examination of the modified cilia of the ear and eye is not feasible, but there is the possibility that they share abnormalities with other ciliated epithelia which can be biopsied. Thus new forms of investigation are opened into conditions of progressive loss of hearing and vision.

The last paragraph of your leader advises caution in the interpretation of electron microscope studies of cilia, with which we wholeheartedly agree; but we would like to point out that the electron-microscopical appearances of abnormal microtubular patterns are so striking that photographic enhancement techniques are unnecessary. More to the point, it is necessary to investigate large samples of material in many patients and controls because of the presence of abnormal microtubular patterns in a proportion of cilia in people who have no disease.

The interest of cell biologists has been aroused by these new reports. In Kartagener's syndrome the clinical observations provided the final confirmation of the essential role of dynein arms in cilial immotility[2]: it may be that the asymmetries recently found in human disease will provide further clues to even more profound biophysical puzzles.

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Diabetic (insulin) oedema

SIR,—A short report on insulin oedema (21 July, p 177) affirmed that oedema of this type is rare in diabetics. We have recently described four patients (three male and one female aged 18-21 years) with typical acute diabetic oedema[1-3] and agree that the severe fluid retention which transiently follows the restoration of good diabetic control is not common. These episodes of oedema were associated with insulin treatment of diabetes, but, in contrast to the case reported by Mr N R Bleach and others, one patient (a boy of 12) had been on insulin for two years. All these episodes of acute oedema were not only transient but settled spontaneously *without diuretic therapy*, which should probably be eschewed in such patients.

We would like to draw particular attention, however, to subacute and chronic forms of oedema occurring in diabetics. This concept was first raised in a presentation to the British Diabetic Association in September 1977 and has been elaborated on elsewhere.[2 3] Over the past few years we have documented 12 diabetics, all female, who suffered from severe generalised fluid retention lasting from a few months to several years. Two of these patients were not treated with insulin and all were observed to have fluid retention with the distribution described by Mr Bleach and his colleagues while on no diuretic therapy. (This latter point is of particular importance in the light of a recent report[4] attributing unexplained oedema to diuretics.) Furthermore, in a survey of 86 insulin-requiring patients under 50 years attending a diabetic clinic, a history of intermittent mild-to-moderate fluid retention without obvious cause was obtained in 14 of 40 (27%) female patients. Only one of 46 male patients gave such a history. Subacute and chronic forms of diabetic oedema are therefore not uncommon and this diagnosis should be considered in female diabetics with unexplained fluid retention. Clinically such diabetic oedema resembles the syndrome of idiopathic oedema of women where there is a high familial incidence of diabetes.[2 5 6]

With regard to the aetiology of diabetic oedema, other possibilities coexist with the mechanisms mentioned by Mr Bleach and his colleagues. Acute diabetic oedema has been considered to resemble "refeeding" oedema[7] and thus to represent a pathological degree of carbohydrate-induced antinatriuresis.[8 9] An abnormal degree of sodium retention, not always manifest as oedema, occurs not only in carbohydrate refeeding but also follows the treatment of ketoacidosis in man[10] and animals.[11]

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Royal Commission report

SIR,—Your leading article on the report of the Royal Commission on the National Health Service (28 July, p 227) waxed lyrical about the preventive content of the proposals. As a trainee in community medicine I beg to differ.

Certainly the commitment is there (paragraph 5.1.): ". . . we regret that more emphasis has not been placed in the past on the preventive role of the NHS. This must change if there are to be substantial improvements in health in the future. . . ." But what "change" is recommended? The answer is very little. Of the six preventive recommendations, four relate to health education (one of the least effective means of prevention), one (on screening) has preoccupied the DHSS for years, and the other (on seat belts) is no stranger to the House of Commons, which may finally legislate on the subject this session.

The most frustrating feature of the report is the total failure of the commissioners to harness their preventive philosophy (which is admirable in conception if not in application) to an existing organisational framework of implementation—namely, community medicine. For prevention is the raison d'être of community medicine, a point which may not have been sufficiently impressed on the commissioners by the representatives of the specialty when evidence was submitted. The commissioners' pious declaration that "We ourselves believe that the specialty has a future" (paragraph 14.55) carries little conviction when no attempt is made to spell out that future. Indeed, the only recommendation on community medicine, that community physicians should be given adequate supporting staff, merely begs the question "Adequate for what purpose?"

The commissioners have lost an unprece-